

The Effects of Different Times Interval of Physical Activity in Diverse Nutritional Status on Cardio-Vascular Risk Factors in High-Risk Middle Aged Women

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Abstract: Today, cardiovascular diseases are one of the major causes of mobility and mortality in the developed countries. Also, the beneficiary effect of physical activities on risk factors associated with these diseases are well documented, little is known about the engagement time and nutrition status of the subjects in such activities. Therefore, this research was conducted to determine the effects of engagement time under various nutrition statuses in which desirable responses occur. In this study 31 sedentary women who had at least one of the risk factors associated with the cardiovascular diseases were selected as the subjects for this research. They participated in an aerobic exercise program with intensity between 60-70% of their maximum heart rate. The program included 1 h session of exercise, three times a week for 18 weeks. The subjects were randomly assigned into three groups, including the exercise condition 1- in the early morning prior to consuming breakfast; exercise condition 2- fed 5 h following lunch in the afternoon and the control group with no exercise protocol. All the subjects completed the research protocol. Triglyceride, HDL, LDL level of the blood of the subjects as the factors associated with the incidence of cardiovascular disease was measured at 6, 12 and 18 weeks following the start of exercise program. The beneficiary effects of participation in physical activities in the fasting condition in the morning was statistically more significant than the fed group in regard to the level of HDL following the 18 weeks. In addition, the level of blood cholesterol and lipid varied significantly in both exercise groups following 6 and 12 weeks of exercise, whereas these changes were significantly more significant in the fasting group. The level of blood LDL did not change significantly in all groups ($p < 0.05$). Cholesterol, lipids and LDL density variation due to the participation in physical activities is not dependent upon the morning or afternoon engagement. However, the beneficence effects of such activities on HDL level changes may be more pronounced during the fasting status.

Key words: Physical activity, nutritional status, cardiovascular risk factors, middle age women

INTRODUCTION

Cardiovascular disease is the major killer in the United States and most other industrialized nations. Approximately half million US adults suffer from stroke each year (first attack), with 100,000 recurrent attacks (Duttary, 2005; Crouse *et al.*, 1997). Approximately, 160,000 of these cases are fatal (Crouse *et al.*, 1997; Donovan *et al.*, 2005). Hyperinsulinemia, hypertension, dyslipidemia and android-shaped obesity are fundamental risk factors for coronary artery disease. These risk factors have a tendency to aggregate and their combination has been called the metabolic syndrome.

Both clinical data and studies in animal models demonstrate that environmental manipulation may affect some of the biochemical indicators associated with the

metabolic syndrome (Crouse *et al.*, 1995; Hubinger and Mackinnon, 1996; Kiens and Litnell, 1982). Participation in regular exercise pursuits is highly recommended for prevention of coronary artery disease (Haskell, 1984; Rauramaa *et al.*, 1984; Regina *et al.*, 2006; Wood *et al.*, 1985). Among the major mechanism by which regular physical activity and exercise training mitigate the risk of coronary heart disease is its impact on high-density lipoprotein metabolism (Leon *et al.*, 1979; Lindgren *et al.*, 1969). Both acute and chronic exercises are reported to have triglyceride-lowering effects (Leon *et al.*, 1979; American Heart Association, 2000; Haskell, 1986; Lemon, 1991; Leon and Sanchez, 2001). It has been shown that exercise may have a smaller effect on plasma total Triglyceride (TAG) in subjects who have relatively low triglyceride levels (Despres and Lamarch, 1989) therefore, exercise-induced lowering.

Repeated angiographies have indicated that there is a direct association between the plasma density reduction and decrease of atherosclerosis progress. One study reported that one-percent decrease in the level of cholesterol was associated with a two percent decline in the incident of coronary heart disease (Giada *et al.*, 1991). In the other hand, there are evidences indicating that some by-product of cholesterol are carcinogenic and lead to mutation. About the effects of physical activities on blood lipids and lipoprotein, it should be mentioned that the majorities of the studies have been conducted for a short duration between 4 to 10 weeks (Frank *et al.*, 1995; Leaf and Kuche, 1988; Wood and Marcia, 1990). However, in longer duration studies for determining the effects of physical activities on blood lipids and lipoprotein profile, conflicting findings have been reported (Janson *et al.*, 2003; Yen and Tasi, 1995). More researches that are recent have examined the nutrition status of the individuals and its effect on risk factors, enzyme activities and physiological variables (Haskell, 1984; American College of Sports Medicine, 1990; Bielinski *et al.*, 1985; Krauss and Burke, 1982; Schwartz *et al.*, 1991). This is despite the fact that in majorities of these studies, high calorie diet or consumption of some of fatty acid without participation in any kind of physical activities were examined (Schwartz *et al.*, 1991; Oyelola *et al.*, 1993).

However, research for comparing the effect of daytime participation in physical activity on the level of blood lipids and lipoproteins are rare (Tran *et al.*, 1983). Therefore, the present research was conducted to evaluate the time interval of physical activities under various nutrition statuses on cardiovascular risk factors in middle age women with such condition. The time interval evaluating the effects of physical activity was the early morning prior to the eating breakfast (fasting) versus the afternoon time (Fed).

MATERIALS AND METHODS

Subjects: Forty two middle age women between the ages 40-55 years voluntarily participated in this study. The subject learned about the study through advertising in shopping centers in the city of Tehran. They had at least one of the cardiovascular risk factors such as excess weight, menopause, mild hypertension or sedentary life style. All subjects have no experience of sport activities within the last three years. The subjects completed a questionnaire in which a complete history for cigarette smoking habit, kidney, liver, gastrointestinal diseases in addition to treatment with beta blockade and/or other medicine consumption to reduce blood pressure or plasma lipids was examined. Following the inspection of the

completed questionnaires, 31 women were nominated for the study. After the explanation of the procedure for study participation, eight of them refused to participate in the protocol but agreed to serve as the control group. The remaining 23 subjects were randomly assigned into two groups of 12 and 11 persons. Again, a complete explanation of the protocol was given to the subjects reminding them for their commitment to follow the instructions for the diet.

Protocol and procedures: Both experimental groups participated in an exercise program purposed by ACSM (Berg *et al.*, 1994) for 18 weeks at 60-70% of maximum heart rate, three sessions a week. The activities included brisk walking or jogging performed on a flat surface in a stadium. The experimental group-1 participated in the exercise program before having breakfast (6: 00 AM) and following 10 h of fasting. The experimental group-2 took part in the exercise program in the afternoon- 3-5 h after eating a complete meal (17: 00PM).

Diet and physical activity record: The range for target heart rate for each subject was calculated by the following equation:

$$\text{Target heart rate} = [(220 - \text{age}) (60 - 70\%)]$$

The heart rate was measured using a polar heart rate monitor (polar electro Inc., Woodbury, NY) during the physical activities.

Subjects completed an initial 3-d dietary record which was analyzed to determine baseline total caloric intake and nutrient composition.

Blood sampling: Blood sampling from each subjects were collected following a minimum of 12th fasting and at approximately the same time of day under both experimental conditions. Blood samples were also collected at four time points for each exercise condition: 24 h before the physical activities and after 6, 12, 18 weeks of training. In an attempt to avoid the known circadian rhythm in blood lipid and lipoproteins (Seip *et al.*, 1993) all samples from both groups were procured within 60 min of each other (between 06 : 00 -07:00 h). Blood sample were obtained in a seated position after 5 min of seated rest from an antecubital vein using a vacutainer system to draw blood in to 2 mL tubes containing no additives and immediately placed on ice. Serum was obtained by centrifugation at 1500 g for 15 min and serum sample were subsequently stored at -70°C for future analysis. All samples for the lipid and lipoprotein analysis were frozen

according to the instruction of the manufacturers of the kit and then batch analyzed on the same day, using the same kit.

Lipid concentrations: HDL-C was isolated prior to frozen storage of serum by the method of Warnick and Gidez. HDL-C and total serum cholesterol were then analyzed using the color metric enzymatic determination methods of Allain and Grande. Cholesterol values were estimated using a Raichem cholesterol reagent diagnostic kit (Raichem, Div. Of Hemagen Diagnostics, Inc., San Diego, CA). TG concentrations were estimated using the enzymatic methods of Bucolo and David (Chang *et al.*, 2003).

A Raichem triglyceride GPO reagent diagnostic kit was used to estimate triglyceride concentrations. All samples were analyzed in triplicate and each subject's samples were analyzed within the same run for each measure. The inter-and intra-assay CV was <4.3%. The TC, HDL-C and plasma TG concentration were used to estimate LDL-Cholesterol (LDL-C) by means of the Friedewald equation as follow:

$$\text{LDL-C} = [\text{TC} - (\text{plasma TG}/2.2) + (\text{HDL-C-TC})]$$

Statistical analysis: Following the completion of the project, statistical analysis was performed on data. Kolmogorov-smirnov test was used to check the normality of distribution of the variables. After normality was conformed, Analysis of variance-repeated measure was employed to analyze the changes across the time (weeks) and ANOVA was used to compare the experimental groups at the base line, 6, 12 and 18 weeks of exercise. Statistical significance was set at $p \leq 0.05$ for all statistical tests.

RESULTS

The characteristics of the subjects are presented in Table 1. The changes in the levels of the dependent variables including TG, TC, HDL and LDL-C are presented in Table 2. While the decrease of TG was statically significant in the fasting group following the 6 weeks ($p = 0.010$) and 12 weeks ($p = 0.017$), this change was not significant ($p = 0.10$) after 18 weeks of exercise. The concentration of TG in the feed group following the 6 and 12 weeks of exercise increased significantly ($p = 0.003$) and ($p = 0.009$), respectively but this change was not significant at the end of the 18 weeks comparing to the start of the study. The change in the concentration of TC in the fasting group following the 6 and 12 weeks was statistically significant ($p = 0.001$), ($p = 0.030$), respectively.

Table 1: Subjects baseline descriptive data

Variable	FA	FE	CON
No. of Participants	12	11	8
Age (year)	45.1±4.32	47.1±6.41	47.2±5.33
Height (cm)	164±5.08	167±3.4	161.5±6.43
Weight (kg)	70.12±12.04	78.42±10.52	64±16.8

All values are mean±standard deviation

Table 2: Blood lipid changes

Variable	Baseline	6 weeks	12 weeks	18 weeks
TG				
FA	110.58±37.14	99.58±31.41*	94.83±29.59*	89.08±27.03
FED	117.63±50.51	105.09±41.96*	94.54±33.31*	96.00±30.70
CON	115.87±33.48	117.37±37.86	121.00±38.84	119.12±36.02
TC				
FA	155.50±9.12	142.33±8.04*	132.45±7.66*	133.29±7.22
FED	129.81±28.98	116.45±27.63*	106.31±24.28*	103.63±21.88
CON	148.21±26.28	150.86±33.87	155.00±33.47	151.50±31.86
HDL-C				
FA	40.25±3.41	40.33±3.77	40.91±4.23	43.33±5.89*
FED	43.18±7.02	43.59±6.71	44.40±6.80	44.54±7.59
CON	45.00±4.78	44.75±5.72	45.25±5.80	44.50±6.04
LDL-C				
FA	100.54±9.11	100.50±8.97	99.83±6.80	98.66±9.46
FED	102.00±9.69	101.81±9.83	103.81±12.63	103.63±8.60
CON	118.86±19.32	119.12±19.98	113.12±12.92	112.75±16.35

*Indicates significant difference between condition. All blood lipid concentration is reported as mg/dl (means±standard deviation). Means with the same lower case superscript are similar within condition ($p > 0.05$)

However, the changes of TG concentration following the 18 weeks of exercise was not significant in both experimental groups ($p = 0.57$). HDL-C concentration increased significantly in the fasting group after final measurement (18 weeks), ($p = 0.001$). The variation in the concentration of LDL-C was not statistically significant in all the stages of exercise protocol. Also, no significant change was found in the control group following the 18 weeks of the research project ($p > 0.05$).

One-way analysis of variance and LSD test results indicated that there was only significant difference in LDL at the base line between the experimental group and the control group ($p < 0.05$). Similar analysis was employed after 6 weeks for all variables. The results showed that there was significant difference between the level of LDL-C and cholesterol of the experimental groups versus the control group ($p < 0.05$). Also, for comparing the results of the experiment after 12 weeks, the same procedure was used. The results of one-way ANOVA and LSD post hoc test showed that the concentration of cholesterol and TG level was significantly different in three groups ($p < 0.05$). At this stage, the difference in cholesterol level was between the feed and fasting groups and between these two groups and the control group ($p < 0.05$). Finally, the results of similar analysis performed on the level of cholesterol and LDL after 18 weeks showed that there was significant differences between the fasting and fed groups and also between the fed and control group in regard to the cholesterol level ($p < 0.05$) and between the LDL level of the fed versus the fasting group ($p < 0.05$).

DISCUSSION

The findings of the present research indicate that the concentration of blood TG in the fasting and fed groups following the 6 and 12 weeks of physical activities changed significantly, but these changes were not significant after 18 weeks of exercise. In this research, the intensity and volume was purposed (Kiens and Lithell, 1982). Considering the sufficiency of the volume for exercise and lack of increase in extra energy expenditure in the subjects throughout the research project, it seems that the program could result in the decrease in the blood TG level. In most research reported, the level of TG following participation in physical activities showed decrease (29, 38, 39) (Krauss and Burke, 1982; Kin Isler *et al.*, 2001; Lampman *et al.*, 1980).

It has been demonstrated that when the initial level of TG prior to the participation in physical activity program is high, the exercise program will lead to more decrease in the TG plasma level.

However, in regard to the absence of significant changes in TG density following the 18 weeks comparing to the starting stage prior to the exercise program, it should be noted that in individuals whose blood TG level is below 120 mg dL^{-1} , exercise generally will not reduce it significantly (Hubinger and Mackinnon, 1996) and what was observed in this research also confirmed this finding. It was also expected that the lipid changes in the fasting group continued to the end of the research project since the decrease of carbohydrate storage in fasting on prolonged exercise exposure lead to increase in the oxidation of the lipid during the rest following the exercise. Finally, in regard to the decrease in TG density, it is purposed that in the post exercise state, the absolute as well as the relative contribution of lipid to energy metabolism has been shown to be increased (Lithell *et al.*, 1984).

The findings of this research show that the blood TG level of the subjects following 6 and 12 weeks of physical activity in both the fasting and fed group decreased significantly. The existing literature in this regard present evidences that show participation in aerobic exercises decreases the level of cholesterol (Leaf *et al.*, 1988; Dufaux *et al.*, 1986; Razz *et al.*, 1988). In was also shown in a research that exercise performed to 75% of maximum oxygen consumption resulted in similar responses in the cholesterol density (Eliakim *et al.*, 2000). In this research, the significant changes in cholesterol level were similar in all time intervals and nutrition status had no effects on this result. Probably, by controlling the relative calorie expenditure, the effects of physical activity under the various nutrition statuses on blood TC level is similar.

The lack of significant decrease in TC level in 12-18 week intervals may be attributed to the insufficiency of intensity of exercise volume and increase in the level of physical fitness of the subjects. Finally, the changes in TC are consistent with the notion that exercise can favorably alter the lipid markers of cardiovascular disease risk by augmenting the reverse cholesterol transport pathway (Lindgren *et al.*, 1969). Very active middle aged men and women show lower plasma concentration of TC in active compared to inactive individuals (Lindgren *et al.*, 1969).

The research findings indicated that blood HDL density in the fasting group and following the 18 weeks of exercise increased significantly. In some researches, it has been reported that the concentration of HDL following the aerobic exercise increased (Kin Isler *et al.*, 2001; Westcott *et al.*, 2001). In the other hand, in one research, no increase in the level of HDL even 24 weeks after exercise at 50-80% of maximum oxygen consumption was found (Schwartz *et al.*, 1991) whereas in one research, the effects of combing diet on plasma HDL level was reported (Murphy, 2000).

Although such changes in these lipid and lipoproteins are likely to be related to the total energy expenditure, there is insufficient evidence to decide whether energy expenditure, intensity of effort, or some combination is responsible (Bucolo and David, 1973).

The mechanisms by which this reverse cholesterol aerobic exercise may influence blood lipid profiles is by modifying the activities of intravascular enzymes and transfer proteins (Haskell, 1984; Leaf *et al.*, 1988). Elevations in the activity of Lipoprotein Lipase (LPL_a) and Lecithin, Cholesterol Acyltransferase (LCAT_a), have been shown after both exercise training (Chang *et al.*, 2003) and after 5 days of military field training (Murphy, 2000). In addition, a reduction in the concentration of Cholesterol Ester Transfer Protein (CETP), which is closely related to CETP activity (CETP_a), has been demonstrated after exercise training.

Greater LPL_a or LEA_a brought about by exercise training may reduce TG concentration and facilitate an increase in HDL-C (Thompson *et al.*, 1982). Similarly, an exercise induced suppression of hepatic TG lipase activity or CETP_a may slow the catabolism of HDL particles by enhancing the accumulation of cholesterol in all HDL sub fraction (Giada *et al.*, 1991; Cefalu, 2001). In addition, 2 potential mechanisms may explain the different change in the lipid/lipoprotein metabolism in different status. Firstly, common postulation is that a threshold for energy expenditure, rather than a specific exercise intensity or duration, may be critical for inducing changes in HDL-C (Hevener *et al.*, 2000). The energy threshold seems to

vary directly with the functional capacity of the subject (Despres and Lamarche, 1989). For example, increased carbohydrate ingestion can reduce LPL_a and HDL-C and increase TG concentration. Conversely, increased cholesterol or fat intake can elevate cholesterol concentrations in all the lipoprotein fractions (Lampman *et al.*, 1980; Pate *et al.*, 1995). According to these cases, probably physical activity in hunger or fasting is one of the definite causes of observing of such results.

The findings of this research also demonstrated that the changes of LDL-C concentration in all groups were not statistically significant throughout the experiment. Such findings have also been reported in other researches (Haskell, 1984; Durstine and Haskell, 1992). In some researches in regard to evaluating the effects of physical activities on LDL-D, the sub-components of this blood parameter is measured (Tran *et al.*, 1983; Thompson *et al.*, 1982). In this research, regular physical activities and weight reduction have increased the LDL peak flotation rate and LDL peak particle diameter following a short period of exercise participation and decreased the concentration of small LPL_s of plasma, while on the average level, no change of the LDL-C in plasma is observed. It was purposed that change of LDL components and its distribution since these two factors lead to simultaneous changes in small, medium and large LDL_s (Leaf *et al.*, 1988). These results have significant application, because it has been shown that the presence of high level of small particles in plasma is associated with increased risk of coronary heart diseases (Blair *et al.*, 1983). Therefore, the lack of any change in plasma LDL-C level following exercise usually can not be an indication for the lack of desirable change in subunits of LDL-C. Another significant factor that may be considered in explaining the contradictory results in different studies is the initial condition of lipoproteins (Reseland *et al.*, 2001). In fact, the more the initial condition is far from the normal, the more significant the response to the exercise may be. In addition, it should be pointed that the majority of the researches in which moderate to high intensity exercise is employed to evaluate the effects of such activities in lipoproteins have done so for a short period of time (Stephen *et al.*, 1997). Therefore, the energy deficiency induced by exercise in the majority of these researches may not have been sufficient for causing significant improvement in plasma lipoprotein level. Finally, it is evident that even exercise under various nutrition statuses could not lead to desirable change in LDL-C level.

REFERENCES

- American Heart Association, 2000. Heart and stroke statistical update. Dallas, Tex: American Heart Association.
- American College of Sports Medicine, 1990. The recommended quantity and quality of exercise for developing and maintaining cardio respiratory and muscular fitness in healthy adults. Med. Sci. Sports Exercise, 22: 265-274.
- Asim, K., Duttaroy, 2005. Postprandial activation of haemostatic factors: Role of dietary fatty acid. Prostaglandins, Leukotrienes and Essential Fatty Acids, 72: 381-391.
- Berg, A., I. Frey, M. Baumastark, M. Halle and J. Keul, 1994. Physical activity and lipoprotein disorders. Sports Med., 17: 6-21.
- Bielinski, R., Y. Schutz and E. Jéquier, 1985. Energy expenditure during the post exercise recovery in man. Am. J. Clin. Nutr., 42: 69-82.
- Blair, S.N., K.H. Cooper, L.W. Gibbons, L.R. Gettman, S. Lewis and N. Goodyear, 1983. Changes in coronary heart disease risk factors associated with increased treadmill time in 735 men. Am. J. Epidemiol., 118: 352-359.
- Bucolo, G. and H. David, 1973. Quantitative determination of serum triglycerides by the use of enzymes. Clin. Chem., 19: 476-482.
- Cefalu, W.T., 2003. Insulin resistance: Cellular and clinical concepts. Exp. Biol. Med., 226: 13-26.
- Chang Do Lee, Aaron, R. Folsom and S.N. Blair, 2003. Physical activity and stroke risk: A meta-analysis. Stroke, 34: 2471-2481.
- Crouse, S., B. O'Brien, J. Rohack, R. Lowe, J. Green, H. Tolson and J. reed, 1995. Changes in serum lipids and apolipoproteins after exercise in men with high cholesterol: Influence of intensity. J. Applied Physiol., 79: 279-286.
- Crouse, S. *et al.*, 1997. Effects of training and a single session of exercise on lipids and apolipoproteins in hypercholesterolemic men. J. Applied Physiol., 83: 2019-28.
- Despres, J.P. and B. Lamarche, 1989. Low-intensity endurance exercise training, plasma lipoproteins and the risk factors : A cross-sectional study. JAM. Med. Assoc., 262: 2395-2401.
- Donovan, G., A. Owen, S.R. Bird, E.M. Kearney and A.M. Nevill *et al.*, 2005. Changes in cardiorespiratory fitness and coronary heart disease risk factors following 24 weeks of moderate-or high-intensity exercise of equal energy cost. J. Applied Physiol., 98: 1619-1625.

- Dufaux, B., U. Order, R. Muller and W. Hollmann, 1986. delayed effects of prolonged exercise on serum lipoproteins. *Metabolism*, 35: 105-109.
- Durstine, J.L. and W.L. Haskell, 1992. Effects of exercise on plasma lipid and lipoproteins. *Exercise Sport Sci. Rev.*, 22: 477-521.
- Eliakim, A., G.S. Makowski, J.A. Brasel and D.M. Cooper, 2000. Adiposity, lipid levels and brief endurance training in nonobese adolescent males. *Int. J. Sport. Med.*, 21: 332-7.
- Frank, J.S., A.D. Watson and P.A. Edwards, 1995. Pathogenesis of atherosclerosis. *Am. J. Cardiol.*, 76: 18-23.
- Giada, F. *et al.*, 1991. Specialized physical training programs: Effects on serum lipoprotein cholesterol, apolipoprotein A-I and B and lipolytic enzyme activities. *J. Sports Med. Phys. Fitness*, 31: 196-203.
- Haskell, W.L., 1986. The influence of exercise training on plasma lipids and lipoproteins in health and disease. *Act. Med. Scand. Supple.*, 711: 25-37.
- Haskell, W.L., 1984. The influence of exercise on the concentration of triglyceride and cholesterol in human plasma. *Exercise Sport Sci. Rev.*, 12: 205-244.
- Hevener, A.L., D. Reichart and J. Olefsky, 2000. Exercise and thiazolidinedione therapy normalize insulin action in the obese Zucker fatty rat. *Diabetes*, 49: 2154-2159.
- Hubinger, L. and L.T. Mackinnon, 1996. The effect of endurance training on lipoprotein (a) [Lo (a)] levels in middle-aged males. *Med. Sci. Sports Exe.*, 28: 757-764.
- Janson, M., R. Gill and E. Adriann, 2003. Hardman. Exercise and postprandial lipid metabolism: An update on potential mechanisms and interaction with high-carbohydrate diets (review). *Nutr. Biochem.*, 14: 122-132.
- Kiens, B. and H. Lithell, 1982. Lipoprotein metabolism influenced by training-induced changes in human skeletal muscle. *J. Clin. Investigation*, 54: 107-112.
- Kin Isler, A., S.N. Kosar and F. Korkusuz, 2001. Effects of step aerobics and aerobic dancing on serum lipids and lipoproteins. *Sports Med. Phys. Fitness*, 41: 380-5.
- Krauss, R.M. and D.J. Burke, 1982. Identification of multiple subclass of plasma low density lipoprotein in normal humans. *J. Lipid Res.*, 3: 97-104.
- Lampman, R.M., J.T. Santinga, D.R. Bassett, W.D. Block and N. Mercer, 1980. Type IV hyperlipoproteinemia: Effects of a caloric restricted type IV diet versus physical training plus isocaloric type IV diet. *Am. J. Clin. Nutr.*, 33: 1233-1243.
- Leaf, D.A. and K. Kuehl, 1988. Acute prolonged sub anaerobic exercise on hypertriglyceridemics : Effects of plasma lipids, lipoproteins and respiratory exchange ratio slope. *Ann. Sports Med.*, 3: 2423-2246.
- Lemon, P.W. and R., 1991. Protein and amino acid needs of the strength athlete. *Int. J. Sports Nutr.* 1: 127-145.
- Leon, A.S. *et al.*, 1997. Effects of a vigorous walking program on body composition and carbohydrate and lipid metabolism of obese young men. *A., J. Clin. Nutr.*, 33: 1776-1787.
- Leon, A.S. and O.A. Sanchez, 2001. Response to blood lipids to exercise training alone or combined with dietary interventions. *Med. Sci. Sports Exercise*, 33: 502-515.
- Lindgren, F.T. and L.C. Jensen *et al.*, 1969. Flotation rates, molecular weights and hydrated densities of the low density lipoproteins, 4: 337-44.
- Lithell, H., R. Schele, B. Vessby and I. Jacobs, 1984. Lipoproteins, lipoprotein lipase and glycogen after prolonged physical activity. *J. Applied Physiol.*, 57: 698-702.
- Murphy, S.L., 2000. Deaths: Final data for 1998. *Natl Vital Stat. Rep.*, 48: 1-106.
- Oyelola, O.O. *et al.*, 1993. Plasma lipid, lipoprotein and apoprotein profiles. In: *Nigerion University athletes and non-athletes*. *Br. J. Sports Med.*, 27: 271-4.
- Pate, R.R., M. Pratt, S.N. Blair, W.L. Haskell, C. Macera and A. Bouchard *et al.*, 1995. Physical activity and public health. A recommendation from the centers for disease control and prevention and the American College of Sports medicine. *J. Am. Med. Assoc.*, 273: 402-407.
- Rauramaa, R. *et al.*, 1984. Effects of mild physical exercise on serum lipoproteins and metabolites of arachidonic acid: A controlled randomized trial in middle aged men. *Br. Med. J.*, 288: 603-607.
- Razz, I., H. Rozenbilt and J.P. Kark, 1988. Effect of moderate exercise on serum lipids in young men with low high density lipoprotein cholesterol levels. *Arteriosclerosis*, 8: 245-51.
- Reseland, J.E., S. Snderson, K. Solvoll, I. Hjermann and P. Urdal *et al.*, 2001. Effect of long-term changes in diet and exercise on plasma leptin concentrations. *Am. J. Clin. Nutr.*, 73: 240-245.
- Regina, C. and M. Burneiko *et al.*, 2006. Interaction of hypercaloric diet and physical exercise on lipid profile, oxidative stress and antioxidant defense. *Food Chemical Toxicol.*, 44: 1167-1172.
- Seip, R.L., P. Moulin, T. Cocke, A. Tall, W.M. Kohrt and K. Mankowitz *et al.*, 1993. Exercise training decreases plasma cholesterol ester transfer protein. *Arteriosclerosis Thrombosis*, 13: 1359-1367.

- Schwartz, R.S. and W.P. Shuman *et al.*, 1991. The effect of intensive endurance exercise training on body fat distribution on young and older men. *Metabolism*, 40: 545-51.
- Stephen, F., C. Barbara, C.O.B. Peter, W.G. Robert and C. Lowe *et al.*, 1997. Training intensity, blood lipids and apolipoproteins in men with high cholesterol. *Applied Physiol.*, 82: 270-277.
- Thompson, P.D. *et al.*, 1982. Exercise, diet or physical characteristics as determinants of HDL levels in endurance athletes. *Atherosclerosis J. Sports Med.*, 3: 123-136.
- Tran, Z.V., A. Weltman, V. Glass and D.P. Mood. The effects of exercise on blood lipids and lipoproteins: A Meta analysis. *Med. Sci. Sports Exercise*, 15: 393-402.
- Westcott, W.L., R.A. Winett, E.S. anderson, J.R. Wojcik and R.L. Loud *et al.*, 2001. Effects of regular and slow-speed resistance training on muscle strength. *J. Sports Med. Phys. Fitness*, 41: 154-158.
- Wood, P.D. and M.L. Stefanick, 1990. Exercise, Fitness and Arteriosclerosis. In : Bouchard, C., R.J. Shephard, J. Stephens, J.R. Sttun and B.D. Mepherson (Eds.), *Exercise, Fitness and Health, a Consensus of Current Knowledge*. Human Kinetics Books, pp: 409.
- Wood, P., D. Terry and W.L. Haskell, 1985. Metabolism of substrates: Diet, lipoprotein metabolism and exercise. *Fed. Proc.*, 44: 358-363.
- Yen, G.C. and L.J. Tasi, 1995. Cholesterol removed from lard-water mixture with β -cyclodextrin. *J. Food Sci.*, 60: 561-564.